The impact of reflex creeping in Vojta therapy on locomotion and postural control

Wpływ odruchowego pełzania w terapii Vojty na kontrolę lokomocji i postawy

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Abstract

Vojta therapy is an intervention method mainly applied to infants at risk of cerebral palsy, and many studies have demonstrated the effectiveness of Vojta therapy. This review describes the concept and method of reflex creeping in Vojta therapy. Additionally, this focuses on the mechanisms through which brainstem activation via reflex creeping affects locomotion and postural control. Proprioception stimulus input during reflex creeping in Vojta therapy affects the pontomedullary reticular formation of the brainstem and eventually affects the activation of the central pattern generator and propriospinal neurons through the reticulospinal tract. It can also induce antigravity movements through the lateral vestibulospinal tract. Reflex creeping in Vojta therapy is an intervention that unconsciously improves locomotion and postural control. The proprioception through the reflex creeping in Vojta therapy can improve posture and locomotion by activating the pontomedullary reticular formation of the brainstem.

Streszczenie

Terapia Vojty jest metodą interwencyjną stosowaną głównie u niemowląt zagrożonych mózgowym porażeniem dziecięcym. Wiele badań wykazało skuteczność terapii metodą Vojty. Niniejsza praca poglądowa opisuje koncepcję i metodę odruchowego pełzania w terapii metodą Vojty. Szczególnie koncentruje się na opisie mechanizmów, poprzez które aktywacja pnia mózgu w odruchowym pełzaniu wpływa na ruch i kontrolę postawy. Stymulacja proprioceptywna podczas odruchowego pełzania w terapii Vojty wpływa na tworzenie mostowo-rdzeniowej struktury siatkowej pnia mózgu i ostatecznie skutkuje aktywacją ośrodkowego generatora wzorców i neuronów rdzeniowych przez drogę siatkowo-rdzeniową. Odruchowe pełzanie wywołuje również ruchy antygrawitacyjne przez boczną drogę przedsionkowo-rdzeniową. Odruchowe pełzanie w terapii Vojty to interwencja, która bez udziału świadomości poprawia kontrolę lokomocji i postawy ciała. Stymulacja proprioceptywna w odruchu pełzania metody Vojty może poprawić postawę i lokomocję, aktywując mostowo-rdzeniową strukturę siatkową pnia mózgu.

Introduction

Vojta therapy was first used by the paediatric neurologist Vaclav Vojta in 1959 for children with motor alterations and for infants with a risk of cerebral palsy [1, 2]. Dr. Vojta described reflex creeping and rolling [3] and found functional changes through proper positioning and stimulation of appropriate trigger zones [4, 5]. Stimulation of trigger zones causes tonic muscle contraction patterns on both sides of the neck, trunk, and limbs to induce stereotypical motor responses, leading to improvement in postural control [6–8].

Reflex creeping in Vojta therapy stimulates proprioception the most [5, 9, 10], and proprioception is transmitted by 2 mechanisms (direct and indirect pathways). In the indirect pathway, stimulation of the Ia, Ib, and II fibers ascends to the cerebellum through activation of the spinocerebellar tract and travels from the cerebellum through the thalamus to the cortex. In the direct pathway, impulses from the periosteum, articular membrane, and joint pass through the dorsal fasciculus, medial lemniscus, and thalamus and then up to the cortex [4]. In addition, in the spinoreticular tract, Ia, Ib, and II fibers pass through the posterior horn of the spinal cord and the pontomedullary reticular formation (PMRF) to the thalamus [11] (Figure 1). It has been reported that the application of such periph-
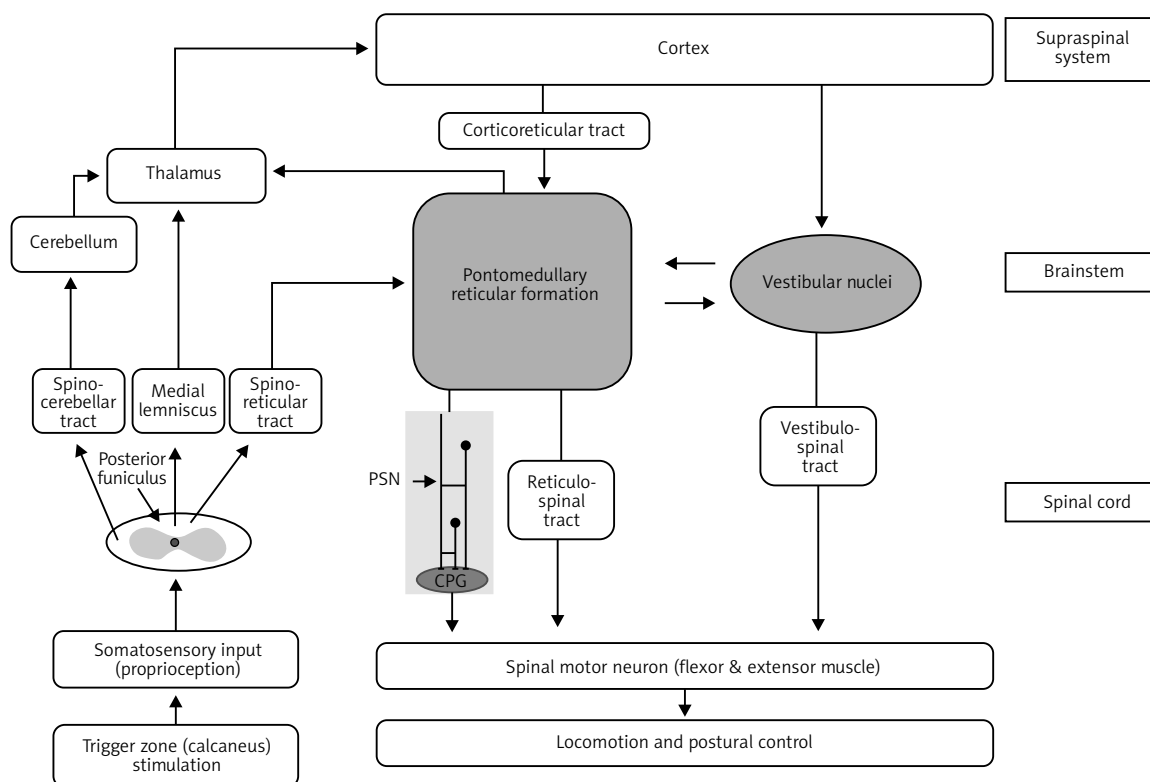
Figure 1. Summary of mechanisms in the brainstem and spinal cord in reflex creeping in Vojta therapy

CPG – central pattern generator, PSN – propriospinal neuron.

eral afferent stimulation generates neuromodulatory activity in subcortical structures such as the cortex, brainstem, cerebellum, or reticular formation [6, 12]. Stimulation of these trigger zones eventually induces unconscious movements in the brain [13], making these stimulations important components for improving or recovering motor function [14–16].

Vojta therapy has been clinically applied in many countries for decades, and its effectiveness has been confirmed [14–20]. Recently, attempts have been made to elucidate the mechanism of Vojta therapy using magnetic resonance imaging (MRI), diffusion tensor imaging (DTI), and electromyography [7, 12, 15, 21, 22]. However, studies on this mechanism have not been systematically established, despite continuous research [13, 21]. Therefore, based on the latest research, we attempted to understand the neurological mechanism of reflex creeping and to prove its effectiveness. Finally, we suggest directions for future research on the neurological mechanisms of reflex creeping.

Description of Vojta therapy

Principle of Vojta therapy

Vojta therapy is also called "reflex locomotion" because it activates involuntary motor response patterns by inducing reflex motor patterns based on stimula-

tion through peripheral pressure [23]. Vojta therapy stimulates the brain through proprioceptive stimulation applied to specific trigger zones in a specific posture, and a complete motor pattern appears as a result of the central nervous system response [14]. The 3 principles applied in Vojta therapy are as follows: 1) automatic postural control, 2) upright posture against gravity, and 3) task-oriented movement of the limbs [1, 2, 24, 25]. Individuals require plans and programs ("innate patterns") that combine task-related automatic adjustments of movement and posture to achieve postural control [1, 13]. In Vojta therapy, motor skills, such as sitting, crawling, and walking, are not trained. Innate movement patterns are activated without conscious intention [13]. Vojta therapy can be applied to a variety of conditions: cerebral palsy, central coordination disorder, peripheral paresis, spinal bifida, scoliosis, musculoskeletal disorders, etc. In addition, it has been proven effective regardless of age. However, patients with fever, children with epilepsy, and children who are extremely sensitive to tactile stimulation should be approached with caution [25].

Application of reflex creeping

Reflex creeping is initiated from the prone position, and the head is rotated 30° to one side so that

Table 1. Trigger zones of reflex creeping and direction of stimulation

Trigger zones on the limbs		Trigger zones on the trunk	
Zone	**Direction**	**Zone**	**Direction**
Lateral process of calcaneal tuberosity	Ventral, cranial, medial/lateral	Trunk	Ventral, caudal, medial
Medial epicondyle of femur	Dorsal, cranial, medial	Gluteus medius	Ventral, cranial/caudal, medial
Radial styloid process	Dorsal, cranial, lateral	Acromion	Dorsal, caudal, medial
Medial epicondyle of humerus	Dorsal, caudal, medial	Anterior superior iliac spine	Dorsal, caudal, medial
		Medial border 1/3 of scapula	Dorsal/ventral, cranial, lateral
		Breast	Dorsal, cranial, medial

the forehead touches the floor. For the arm on the facial side, the shoulder joint is at 125°–130° flexion and 30° abduction, and the elbow joint is flexed at 45° so that the medial epicondyle of the humerus touches the floor. For the occipital leg, the hip joint is at 30° flexion, external rotation, and the knee joint at 45° flexion. The arm on the occipital side is positioned freely along the trunk. The hand and finger joints are also freely positioned. The leg on the facial side is positioned straight from the ground [4, 5]. The trigger zones are the medial epicondyle of the humerus and the calcaneus tubercles. Other trigger zones may also be applied to induce a motor response. The trigger zones and directions of the reflex creeping are listed in Table 1. Thus, it is possible to elicit a better motor response through temporal and spatial summation.

Motor response of reflex creeping

The starting position is essential for eliciting a complete motor response [4, 9]. The global pattern of reflex creeping aims to assume an upright posture achieved through the sequential activation of muscles on both sides of the body. Corresponding points of the extremities (punctum fixation), the elbow of the facial side, and the heel of the occipital side become support points alternately [5].

When stimulated, the trunk is lifted off the floor with the support of the elbow and calcaneus, and the trunk moves forward in the direction of the supporting elbow. The wrist on the facial side is extended, and the fingers are slightly bent. The ankle on the occipital side is supported by maintaining a neutral position. The facial leg is flexed and raised to create a 3-point support, and extension of the spine appears (Figure 2) [4, 5, 26]. Achieving the proper curvature of the spine determines the upright posture of the body and, ultimately, alternating locomotion [2, 5].

Evidence that reflex creeping affects locomotion and postural control

Effects of reflex creeping on pontomedullary reticular formation

The motor response induced by stimulation of trigger zones is controlled by the midbrain or brainstem region [6, 9]. Vojta (1988) reported that the lowest coordination level of reflex creeping is in the dorsal longitudinal fasciculus located in the brainstem, and the highest coordination level is in the midbrain [4]. Among these, PMRF in the brainstem plays a key role in Vojta therapy [1, 6]. The PMRF receives both crossed and uncrossed inputs from the cortex [11, 27] and is involved in postural control and locomotion regulation [28, 29]. The PMRF is an important hub for sensorimotor integration, allowing the nervous system to properly combine voluntary movements with
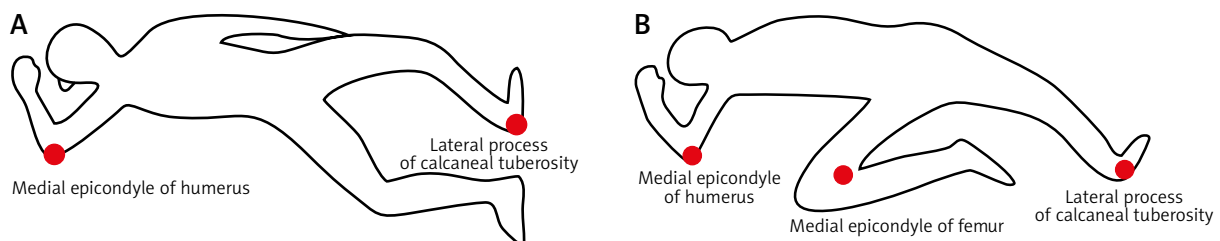

Figure 2. The starting position of the reflex creeping and the posture that appears during the motor response. In the starting position, 2 points (humerus medial epicondyle of facial side and calcaneus of occipital side) are supported (**A**), but 3 points (humerus medial epicondyle of facial side, calcaneus of occipital side, and knee of facial side) are supported during the motor response (**B**)

posture and locomotion [11]. It is also output to the reticulospinal tract and is involved in anticipatory and reactive postural adjustment and the control of muscle tone [11, 28, 30]. Using functional MRI, Hok *et al.* (2017) reported that the PMRF was activated when sequential finger opposition was performed after stimulation of the trigger zone (heel) [6]. Brainstem activation affects unconscious movements of reflex creeping [7]. Ha and Sung (2021) observed changes in the corticoreticular tract (CRT) in children with developmental delays before and after Vojta therapy using DTI [15]. They reported that the tract volume significantly improved, resulting in improved gait and motor function. The CRT terminates in the PMRF, providing volitional input to the PMRF. CRT is involved in generating gait command signals and promoting postural control through the descending reticulospinal tract [31, 32]. Therefore, reflex creeping can affect postural control and locomotion.

Effects of reflex creeping on the central pattern generator

The central pattern generator (CPG) is a neural network capable of independently generating organized patterns of rhythmic motor activity [33, 34]. The CPG plays a role in controlling the motor rhythm that governs human bipedal gait and right-left and flexion-extension patterns of muscle activity [11]. In other words, CPG ensures that the reflexes generated during the gait cycle occur at an appropriate time [35]. Recently, the concept of CPG in humans has been supported because it is known that the brainstem structure is related to human locomotion and postural control [23, 36–38]. Activation of PMRF through trigger zones affects the CPG by activating neuronal circuits in the spinal cord through reticulospinal neurons [11, 38–41]. The CPG induced by reflex creeping produces periodic rhythmic movement patterns [35, 42], which are associated with a stereotypic tonic response induced in response to Vojta therapy [5, 9]. Czajkowska *et al.* (2019) reported that the pressure increased when intraoral pressure during non-nutrition and nutrition was measured before and after Vojta therapy in premature newborns [43]. They reported improvements in sucking regularity and rhythm because Vojta therapy had a direct effect on the sucking CPG. Lim and Kim (2013) found that when reflex creeping was applied to children with cerebral palsy, the CPG was stimulated to activate alternating movements and affect gait function [35].

Activation of the CPG is influenced by propriospinal neurons (PSNs) in the spinal cord [44]. PSNs are a group of spinal intermediate neurons that connect multiple spinal cord segments and participate in complex or long motor reflexes and are known to play an important role in motor control and sensory processing [21, 45]. PSNs play an important role in the plastic organization of spinal circuits and are ideally positioned for motor recovery when supraspinal control is disturbed [21, 46]. PSNs contribute to motor CPG function, where inter-CPG communication or integration of external sensory signals produces rhythmic movements in the extremities [44, 47, 48]. In humans, PSNs participate in a variety of tasks, including the integration/modulation of descending supraspinal pathways (brain commands) and peripheral afferent (transmission of sensory input from receptors) inputs [45, 49]. Gajewska *et al.* (2018) found activation of the deltoid muscle through stimulation of the femoral epicondyle and activation of the rectus femoris through stimulation of the acromion when Vojta therapy was applied to healthy individuals [21]. They concluded that PSNs were activated. Dietz *et al.* (2001) reported that upper extremity muscle (biceps, triceps, deltoid) activity was observed when electrical pulses were applied to the tibial nerve during walking, indicating a nerve connection between the upper and lower extremity muscles [46]. Therefore, stimulation of trigger zones activates PMRF and influences the activation of CPG and PSNs.

Effects of reflex creeping on vestibular nuclei

The vestibular nuclei are interconnected to the supraspinal, brainstem, and spinal cord, which control posture and balance so that vestibulospinal influences are coordinated with the actions of other pathways [11]. The vestibular nuclei receive input from the premotor cortex, somatosensory cortex, cervical spinal cord, proprioceptive system (neck/limb), and visual system [11, 50]. The vestibular nuclei act as a hub for receiving information related to body position. In addition, it is projected onto the oculomotor and trunk muscles, as well as the upper and lower limbs, and plays a role in controlling posture and stabilizing gaze [51]. The vestibular nuclei affect postural control [52, 53] through the lateral/medial vestibular tract and reticulospinal tract [52–55]. The lateral vestibulospinal tract projects to all levels of the spinal cord and forms connections with motor neurons and interneurons [56–58], regulating the activity of α and γ motor neurons in antigravity muscles through intraspinal circuits [11, 53]. Carratalá-Tejada *et al.* (2022) and Epple *et al.* (2020) reported improved postural control after applying Vojta therapy to multiple sclerosis and stroke patients, respectively [1, 14]. Ha and Sung (2021) also reported that postural control improved by tonic muscle contraction after Vojta therapy was applied to children with developmental delay and hypotonia [16]. The head is turned to one side to apply resistance in reflex creeping. As a result, information from the neck is input to the vestibular nuclei and PMRF to estimate self-motion and maintain posture and balance [11]. Information input to the vestibular nuclei through the head affects the antigravity move-

ment of the trunk through the vestibular spinal tract, which is speculated to improve postural control.

Conclusions

Stimulation of the trigger zones of reflex creeping activates the brainstem with proprioceptive inputs to induce unconscious movement. We propose 4 potential areas in which Vojta therapy can influence locomotor and postural control (Figure 1). Future studies on the effects of reflex creeping in Vojta therapy on locomotion and postural control will be required.
1. Reflex creeping affects gait through the PMRF.
2. Reflex creeping affects postural control through the vestibular nuclei.
3. The activation of the PMRF and the vestibulospinal nuclei of the brainstem affects postural control and locomotion.
4. Brainstem activation affects CPG and propriospinal neurons to activate alternating movement.

Conflict of interest

The authors declare no conflict of interest.

References

1. Epple C, Maurer-Burkhard B, Lichti MC, Steiner T. Vojta therapy improves postural control in very early stroke rehabilitation: a randomised controlled pilot trial. Neurol Res Pract 2020; 2: 23.
2. Juárez-Albuixech ML, Redondo-González O, Tello I, Collado-Vázquez S, Jiménez-Antona C. Vojta therapy versus transcutaneous electrical nerve stimulation for lumbosciatica syndrome: a quasiexperimental pilot study. J Bodyw Mov Ther 2020; 24: 39-46.
3. Khan MH, Helsper J, Yang C, Grzegorzek M. An automatic vision-based monitoring system for accurate Vojta-therapy. 2016 IEEE/ACIS 15th ICIS 1-6.
4. Vojta V. Die zerebralen Bewegungsstörungen im Säuglingsalter: Frühdiagnose und Frühtherapi. Ferdinand Enke Verlag, Stuttgart, Germany 1988.
5. Vojta V, Peters A. Das vojta-prinzipmuskelspiele in reflexfortbewegung und motorischerontogenese 3rd ed. Springer MedizinVerlag, Heidelberg, Germany 2007.
6. Hok P, Opavský J, Kutín M, Tüdös Z, Kaňovský P, Hluštík P. Modulation of the sensorimotor system by sustained manual pressure stimulation. Neuroscience 2017; 348: 11-22.
7. Sanz-Esteban I, Cano-de-la-Cuerda R, San-Martín-Gómez A, Jiménez-Antona C, Monge-Pereira E, Estrada-Barranco C, Serrano JI. Cortical activity during sensorial tactile stimulation in healthy adults through Vojta therapy. A randomized pilot controlled trial. J Neuroeng Rehabil 2021; 18: 13.
8. Vojta V. Reflex-Umdrehen als Bahnungssystem der menschlichen Fortbewegung [Reflex rotation as a pathway to human locomotion]. Z Orthop Ihre Grenzgeb 1970; 108: 446-452.
9. Vojta V. Early diagnosis and therapy of cerebral movement disorders in childhood. C. Reflexogenous locomotion: reflex creeping and reflex turning. C1. The kinesiologic content and connection with the tonic neck reflexes. Z Orthop Ihre Grenzgeb 193; 111: 268-291.
10. Vojta V. Early diagnosis and therapy of cerebral motor disorders in childhood. A. Postural reflexes in developmental kinesiology. 3. Pathological reflexes from the aspects of tonic neck reflexes and tonic labyrinthine reflexes. Z Orthop Ihre Grenzgeb 1972; 110: 467-476.
11. Mackinnon CD. Sensorimotor anatomy of gait, balance, and falls. Handb Clin Neurol 2018; 159: 3-26.
12. Sanz-Esteban I, Calvo-Lobo C, Ríos-Lago M, Álvarez-Linera J, Muñoz-García D, Rodríguez-Sanz D. Mapping the human brain during a specific Vojta's tactile input: the ipsilateral putamen's role. Medicine (Baltimore) 2018; 97: e0253.
13. Sanz-Esteban I, Cano-de-la-Cuerda R, San-Martin-Gomez A, Jimenez-Antona C, Monge-Pereira E, Estrada-Barranco C, Garcia-Sanchez PC, Serrano JI. Innate muscle patterns reproduction during afferent somatosensory input with Vojta therapy in healthy adults. a randomized controlled trial. IEEE Trans Neural Syst Rehabil Eng 2021; 29: 2232-2241.
14. Carratalá-Tejada M, Cuesta-Gómez A, Ortiz-Gutiérrez R, Molina-Rueda F, Luna-Oliva L, Miangolarra-Page JC. Reflex locomotion therapy for balance, gait, and fatigue rehabilitation in subjects with multiple sclerosis. J Clin Med 2022; 11: 567.
15. Ha SY, Sung YH. Changes of neural pathways after vojta approach in a child with developmental delay. Children (Basel) 2021; 8: 918.
16. Ha SY, Sung YH. Vojta approach affects neck stability and static balance in sitting position of children with hypotonia. Int Neurourol J 2021; 25: 90-95.
17. Jung MW, Landenberger M, Jung T, Lindenthal T, Philippi H. Vojta therapy and neurodevelopmental treatment in children with infantile postural asymmetry: a randomised controlled trial. J Phys Ther Sci 2017; 29: 301-306.
18. Kiebzak W, Żurawski A, Dwornik M. Vojta method in the treatment of developmental hip dysplasia – a case report. Ther Clin Risk Manag 2016; 12: 1271-1276.
19. Kiebzak W, Żurawski A, Głuszek S, Kosztołowicz M, Białek WA. Cortisol levels in infants with central coordination disorders during Vojta therapy. Children (Basel) 2021; 8: 1113.
20. Sung YH, Ha SY. The Vojta approach changes thicknesses of abdominal muscles and gait in children with spastic cerebral palsy: a randomized controlled trial, pilot study. Technol Health Care 2020; 28: 293-301.
21. Gajewska E, Huber J, Kulczyk A, Lipiec J, Sobieska M. An attempt to explain the Vojta therapy mechanism of action using the surface polyelectromyography in healthy subjects: a pilot study. J Bodyw Mov Ther 2018; 22: 287-292.
22. Hok P, Hlustik P. Modulation of the human sensorimotor system by afferent somatosensory input: evidence from experimental pressure stimulation and physiotherapy. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2020; 164: 371-379.
23. Hok P, Opavský J, Labounek R, Kutín M, Šlachtová M, Tüdös Z, Kaňovský P, Hluštík P. Differential effects of sustained manual pressure stimulation according to site of action. Front Neurosci 2019; 13: 722.
24. Bäckström B, Dahlgren L. Vojta Self-training: experiences of six neurologically impaired people: a qualitative study. Physiotherapy 2000; 86: 567-574 .
25. Bauer H, Appaji G, Mundt D. VOJTA neurophysiologic therapy. Indian J Pediatr 1992; 59: 37-51.

26. Lim H. Comparison of Activity in Scapular Stabilizing muscles during knee push-up plus and modified Vojta's 3-point support exercises. Healthcare (Basel) 2021; 9: 1636.
27. Kably B, Drew T. Corticoreticular pathways in the cat. I. Projection patterns and collaterization. J Neurophysiol 1998; 80: 389-405.
28. Schepens B, Drew T. Independent and convergent signals from the pontomedullary reticular formation contribute to the control of posture and movement during reaching in the cat. J Neurophysiol 2004; 92: 2217-2238.
29. Miller DM, DeMayo WM, Bourdages GH, Wittman SR, Yates BJ, McCall AA. Neurons in the pontomedullary reticular formation receive converging inputs from the hindlimb and labyrinth. Exp Brain Res 2017; 235: 1195-1207.
30. Takakusaki K. Functional neuroanatomy for posture and gait control. J Mov Disord 2017; 10: 1-17.
31. Jang SH, Lee SJ. Corticoreticular tract in the human brain: a mini review. Front Neurol 2019; 10: 1188.
32. Boyne P, Awosika OO, Luo Y. Mapping the corticoreticular pathway from cortex-wide anterograde axonal tracing in the mouse. J Neurosci Res 2021; 99: 3392-3405.
33. Bullock TH. The origin of patterned nervous discharge. Behaviour 1961; 17: 48-59.
34. Wilson DM, Wyman RJ. Motor output patterns during random and rhythmic stimulation of locust thoracic ganglia. Biophys 1965; 5: 121-143.
35. Lim H, Kim T. Effects of vojta therapy on gait of children with spastic diplegia. J Phys Ther Sci 2013; 25: 1605-1608.
36. Jahn K, Deutschländer A, Stephan T, Kalla R, Wiesmann M, Strupp M, Brandt T. Imaging human supraspinal locomotor centers in brainstem and cerebellum. Neuroimage 2008; 39: 786-792.
37. la Fougère C, Zwergal A, Rominger A, Förster S, Fesl G, Dieterich M, Brandt T, Strupp M, Bartenstein P, Jahn K. Real versus imagined locomotion: a [18F]-FDG PET-fMRI comparison. Neuroimage 2010; 50: 1589-1598.
38. Takakusaki K. Neurophysiology of gait: from the spinal cord to the frontal lobe. Mov Disord 2013; 28: 1483-1491.
39. Grillner S. Control of locomotion in bipeds, tetrapods, and fish. Comprehensive Physiology 2011; 1179-1236.
40. Mori S. Integration of posture and locomotion in acute decerebrate cats and in awake, freely moving cats. Prog Neurobiol 1987; 28: 161-195.
41. Rossignol S. Neural control of stereotypic limb movements. Comprehensive Physiology 1996; 173-216.
42. Grillner S, Wallén P. Central pattern generators for locomotion, with special reference to vertebrates. Annu Rev Neurosci 1985; 8: 233-261.
43. Czajkowska M, Fonfara A, Królak-Olejnik B, Michnikowski M, Gólczewski T. The impact of early therapeutic intervention on the central pattern generator in premature newborns – a preliminary study and literature review. Dev Period Med 2019; 23: 178-183.
44. Laliberte AM, Goltash S, Lalonde NR, Bui TV. Propriospinal neurons: essential elements of locomotor control in the intact and possibly the injured spinal cord. Front Cell Neurosci 2019; 13: 512.
45. Jankowska E, Lundberg A, Roberts WJ, Stuart D. A long propriospinal system with direct effect on motoneurones and on interneurones in the cat lumbosacral cord. Exp Brain Res 1974; 21: 169-194.
46. Dietz V, Fouad K, Bastiaanse CM. Neuronal coordination of arm and leg movements during human locomotion. Eur J Neurosci 2001; 14:1906-1914.
47. Gosgnach S, Bikoff JB, Dougherty KJ, El Manira A, Lanuza GM, Zhang Y. Delineating the diversity of spinal interneurons in locomotor circuits. J Neurosci 2017; 37: 10835-10841.
48. Juvin L, Le Gal JP, Simmers J, Morin D. Cervicolumbar coordination in mammalian quadrupedal locomotion: role of spinal thoracic circuitry and limb sensory inputs. J Neurosci 2012; 32: 953-965.
49. Alstermark B, Lundberg A, Pinter M, Sasaki S. Subpopulations and functions of long C3-C5 propriospinal neurones. Brain Res 1987; 404: 395-400.
50. McCall AA, Miller DM, Yates BJ. Descending influences on vestibulospinal and vestibulosympathetic reflexes. Front Neurol 2017; 8: 112.
51. Tighilet B, Chabbert C. Adult neurogenesis promotes balance recovery after vestibular loss. Prog Neurobiol 2019; 174: 28-35.
52. Bach LM, Mogoun HW. The vestibular nuclei as an excitatory mechanism for the cord. Fed Proc 1947; 6: 70.
53. Tanaka H, Nakamura J, Siozaki T, Ueta K, Morioka S, Shomoto K, Okada Y. Posture influences on vestibulospinal tract excitability. Exp Brain Res 2021; 239: 997-1007.
54. Horak FB. Postural compensation for vestibular loss. Ann N Y Acad Sci 2009; 1164: 76-81.
55. Witt EC, Murray AJ. Vestibulospinal contributions to mammalian locomotion. Curr Opin Physiol 2019; 8: 56-62.
56. Grillner S, Hongo T, Lund S. The vestibulospinal tract. Effects on alpha-motoneurones in the lumbosacral spinal cord in the cat. Exp Brain Res 1970; 10: 94-120.
57. Grillner S, Hongo T, Lund S. Convergent effects on alpha motoneurones from the vestibulospinal tract and a pathway descending in the medial longitudinal fasciculus. Exp Brain Res 1971; 12: 457-479.
58. Basaldella E, Takeoka A, Sigrist M, Arber S. Multisensory signaling shapes vestibulo-motor circuit specificity. Cell 2015; 163: 301-312.

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